How Are Genetic Gains Obtained Operationally?

There are two major approaches: the seed orchard approach and clonal forestry. The seed orchard approach involves selecting the best individuals, grafting them in seed orchards, progeny testing the orchard to remove the less desirable clones and providing a new generation to select in. Operational plantations are generated from the seed produced by the orchards.

Clonal forestry depends on the availability of efficient vegetative propagation methods, usually rooted cuttings, sometimes tissue culture. A few highly selected individuals can than be propagated to reforest substantial acreages. Because of the cost involved this is most economical on the best sites, located close to manufacturing facilities. Because the time to deployment is shortened this method lends itself to tailor-making trees for specific products. For example the Aracruz company in Brazil has achieved rotations of 6 to 7 years with eucalypts. With *Gmelina arborea* 4 year rotations are possible. With blocks of well characterized clones, it is possible to fine-tune processing to the individual clones.

See also: **Genetics and Genetic Resources**: Molecular Biology of Forest Trees. **Papermaking**: Overview; World Paper Industry Overview. **Pulping**: Chemical Pulping; Mechanical Pulping. **Tree Breeding, Practices**: Biological Improvement of Wood Properties. **Tree Breeding, Principles**: A Historical Overview of Forest Tree Improvement; Conifer Breeding Principles and Processes; Forest Genetics and Tree Breeding; Current and Future Signposts. **Wood Formation and Properties**: Formation and Structure of Wood.

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Breeding for Disease and Insect Resistance

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Introduction

Pest resistance historically has been the single most important trait in crop breeding, reflecting the vast number of biotic agents that challenge domesticated plants. The number of diseases and insects that afflict forest trees may be even greater than their agricultural counterparts, but no comparable investment to combat them has been made, in spite of the fact that some forest trees have been victims of some of the most spectacular and disastrous epidemics known. Virtual elimination of American chestnut to chestnut blight and extirpation of large populations of American and European species of elm to Dutch elm disease, and white pines to white pine blister rust are textbook examples, as are the depredations of gypsy moth on North American hardwoods. Other important epidemics that started in the last century, some recently, include dogwood anthracnose, butternut canker, Port-Orford cedar root rot, pitch canker of pines, sudden oak death in North America, and the pinewood nematode in Japan. Almost all, of course, are the result of introduced pests. More will undoubtedly follow.

Exotic pests have caused immense economic and ecological damage, and, with few exceptions, are the only ones that merit serious attention. A few pathosystems that exhibit properties of both endemic and exotic diseases are often disturbed, or 'degenerate,' as a result of human intervention (for example, offsite planting, narrow genetic base, dysgenic selection). The same applies to insect pests. The far greater number of endemic forest pests has been regulated by natural selection over epochs of mutual adaptation through coevolution with their hosts.

While much basic understanding of pest resistance and breeding strategies have come from agronomic crops, distinctive properties of tree populations make the former incomplete models. The most important of these properties is the extension of trees in space and time. This has several important biological and practical consequences, especially for disease resistance. Great size projects a tree's parts into different microenvironments above and below ground, providing diversity of niches and habitats,

while great longevity provides a perennial source of energy for different forest organisms, including pathogens and predators. Ontogenetic changes occur in morphology, physiology, and susceptibility to pests. Extension also imposes great logistical constraints to the breeder in time required for trees to reach sexual maturity, as well as access to flowers high in the crown. Great diversity (heterozygosity) of wild tree populations provides an abundant resource of variability, but is accompanied by high genetic load and inbreeding depression. This inhibits use of conventional agronomic breeding tools of selfing and backcrossing, complicating analysis of heritable traits, including resistance.

Mechanisms and Inheritance of Resistance

In comparison with crop pathosystems, relatively little is known about mechanisms and inheritance of resistance in trees to specific pests, especially insects, but several general characteristics are shared, such as hypersensitivity, partial resistance, ontogenetic (age-related) resistance, and tolerance. Morphological traits (e.g., leaf toughness, bark or cuticle thickness, trichomes, hairs) that impede pest feeding or ingress, or phenological traits that put host tissues out of synchrony with the life cycle of the pest are particularly important in host–insect interactions. Constitutive products of secondary metabolism (e.g., phenolics, tannins, monoterpenes) protect against herbivory. Behavioral characteristics (e.g., host preference, apparency, predator attraction) add a dimension of complexity to insect–tree interactions.

Genetic resistance is conditional: it depends as much on the genotype of the pathogen as it does on that of the host. The same is true of pathogen virulence and aggressiveness. Although emphasis is most often placed on host resistance, as if it were independent of the pathogen, it is the interaction phenotype that is inherited, and this is a property of both symbionts. This is made especially clear in gene-for-gene systems.

Hypersensitive reactions (HR) are often controlled by single, dominant, major resistance genes (R genes) in gene-for-gene systems. These systems exhibit precise specificity between interacting gene loci of host and pathogen, in which the resistant interaction phenotype is conditioned by an R allele in the host and a complementary allele for avirulence (AVR) in the pathogen. R alleles function in pathogen recognition and activation of host defenses, often leading to HR. HR causes necrosis in host cells immediately surrounding the lesion, effectively arresting further pathogenesis. The outcome is virtual immunity for the host. Virulence (vR) alleles function to avoid or suppress this recognition, thus genetically restoring a compatible (i.e., susceptible) interaction phenotype. R genes can impose intense selection pressure on pathogen vR genes, which may cause them to increase exponentially in frequency until the usefulness of the R genes deployed in the host population is nullified. However, except when selected for by R genes, vR genes are thought to be less fit than AVR genes, which may explain why they do not become fixed in natural pathosystems. HR and R genes are more likely to be found in specialized, biotrophic pathosystems, such as leaf rusts of poplars and stem rusts of pine. HR is also common in tree–insect interactions, but the genetic basis has not been determined.

Partial resistance (PR), as the name implies, restrains pathogen development and/or reproduction without entirely excluding disease. Epidemiologically, PR functions to reduce the rate of infection, and has often assumed names more descriptive of particular kinds of diseases (e.g., 'slow rusting,' 'slow mildewing'). The degree of protection PR affords ranges widely, but can be highly effective in ensuring survival and mitigating damage. It is usually more complexly inherited than major gene resistance (MGR), and may involve several to many genes. Although much less dramatic than MGR, resistance conferred by PR is likely to be more stable, because it is not vulnerable to pathogen races with specific virulence to it. MGR and PR can exist together and act synergistically.

Ontogenetic resistance (OGR) and tolerance are the least understood mechanisms, but may be the most widespread and important in regulating forest diseases and predators in natural ecosystems. These two mechanisms may represent the greatest contrasts with annual plants in disease interactions. OGR is resistance that increases with age (occasionally decreases, but not to be confused with senescence). It has both a seasonal component, similar to annuals, wherein tissues become morphologically or physiologically less susceptible to pathogenesis or herbivory, and a perennial component that extends over the tree's lifetime. Tolerance implies the ability of a tree to survive, grow, and reproduce despite harboring the pest. Although conceptually clear, it is often difficult in practice to separate tolerance from low levels of resistance.

Unique to woody perennials is the inherent capacity to create barriers that wall off and compartmentalize invading pathogens in living sapwood. Upon injury, parenchyma tissues in rays react, synthesizing phenolic and other toxic compounds

that discolor wood in a zone surrounding the site of infection and confine spread of invading microorganisms to the limits of the reaction zone. Trees also protect living bark from injury from abiotic or biotic origin by formation of a nonsuberized impervious layer of cells in phloem and cortex tissues at the site of the lesion, temporarily isolating invaders from water and nutrients. Necrophyllactic periderms are then laid down behind the injury site which seal the wound. Although both of these reactions are nonspecific, reaction rates are genetically determined and amenable to selection.

General Considerations

Because tree breeding is expensive, there must be a clear economic benefit to justify a program. Usually, only pests of severe epidemic potential will justify the effort, and these are usually ones that have been introduced to a susceptible host population (or vice versa), although in some circumstances, such as widespread off-site planting, dysgenic selection, or crowded monocultures, endemic pests can become epidemic.

There should be some indication that selection and breeding for resistance to the pest will work. Addressing some of the following questions should assist in making a determination:

1. Is there a pattern of infection? Natural stands under strong epidemic pressure from the pest may reveal resistant phenotypes that are unlikely to be chance escapes. Provenance or family trials in established plantations may also show clear genetic differences that can be exploited.
2. Is there an efficient screening technique in place to evaluate candidate parental genotypes? Criteria for resistance should be clear, and expression of the traits selected for unambiguous. Most important, if artificial inoculation/infestation techniques are used, results should be consistent with field performance throughout the length of the rotation.
3. Is the pest more or less specialized in its host range? Usually, there are better chances of finding host genetic variation in resistance to specialists than generalists, and to biotrophs than heterotrophs.
4. Do exotic relatives of the host exist, especially at the pest's gene center, with resistance to the same or related pest? These may be able to be used as resistance donors in interspecific hybridization and backcross breeding.
5. How well is the genetic structure of the pest population known, both in its places of origin and introduction? Is its breeding behavior predominantly outcrossing or inbreeding (or clonal)? The amount of diversity and its potential for recombination will suggest the level of risk to races of wider virulence.

The goal of selection and breeding should always be for durable resistance. R genes, while highly effective in the short term, can be completely overcome by virulence genes with the appropriate specificity. Developing pyramids ('stacking') of different R genes in breeding lines, or buffering MGR with PR, are alternative breeding strategies to prevent or dampen exponential increases in pathogen virulence frequencies.

As detailed an understanding as possible of mechanisms and inheritance of resistance/virulence will always assist in making gene deployment strategies more effective. Nevertheless, resistance can still be used without such knowledge.

A clear distinction must be kept between breeding for production and for information. For example, if it is deemed necessary to understand the inheritance of resistance mechanisms, strict genetic control of both inoculum source and host material must be exercised in an appropriate mating design. If on the other hand the objective is simply to 'pick the winners' with as broad a base of resistance possible, bulk inoculum from throughout the range of intended deployment is more appropriate for screening candidates.

Maintaining as broad a genetic base as possible is probably the most essential requirement of any breeding program. Selection, by definition, narrows the genetic base. But sufficient diversity in either program breeding or archival populations is necessary as a hedge against the risk of new pests arising, or races of the same one with wider virulence. How much diversity is enough is a difficult and controversial issue, and will depend on the specific circumstances. Gene frequencies for resistance to exotic pests can be extremely rare, as for example those that confer resistance to white pine blister rust and root rot of Port-Orford cedar in North America. In such situations, availability of large, wild populations are the best solution, especially if natural selection is being imposed by the pest.

Examples of Active Resistance Breeding Programs

Table 1 lists seven major forest tree disease or insect epidemics that have motivated breeding programs. These include canker diseases, a vascular wilt,

Table 1 Examples of major pests with active breeding programs

Disease/pest	*Host*	*Pathogen/pest*	*Type of disease*	*Origin*	*Type of resistance (parameter)*
Chestnut blight	*Castanea dentata*	*Cryphonectria parasitica*	Canker	Exotic	PR (canker size)
Dutch elm disease	*Ulmus americana*	*Ophiostoma ulmi*, *O. novo-ulmi*	Vascular wilt	Exotic	Tolerance (% crown damage)
White pine blister rust	*Pinus* (subsect. *Strobi*)	*Cronartium ribicola*	Canker	Exotic	MGR, PR (infection frequency; canker abortion, size)
Leaf rusts	*Populus, Salix*	*Melampsora* spp.	Leaf rust	Exotic, Indigenous	MGR, PR (slow rusting)
Fusiform rust	*Pinus taeda, P. elliottii*	*Cronartium quercuum* f.sp. *fusiforme*	Canker	Indigenous (degenerate)	MGR, PR (% infection, canker size)
Port-Orford cedar root rot	*Chamaecyparis lawsonia*	*Phytophthora lateralis*	Root rot	Exotic	MGR?
White pine weevil	*Picea sitchensis*, *P. glauca*	*Pissodes strobi*	Shoot feeder	Exotic, indigenous	PR (% infested)

foliage rusts, a root rot, and a shoot-feeding insect. Thumbnail sketches of problems and progress in four of these follow, illustrating some of the general principles discussed above. Some are also covered in other articles in this volume (*see* **Pathology**: Vascular Wilt Diseases; Leaf and Needle Diseases; Stem Canker Diseases; Pine Wilt and the Pine Wood Nematode; Rust Diseases; Insect Associated Tree Diseases).

Chestnut Blight

Chestnut blight is responsible for one of the worst epidemics of trees, if not all plants, in history, having destroyed the dominant species (*Castanea dentata*) of an entire ecosystem that reached from Maine to Georgia. Caused by *Cryphonectria parasitica*, introduced from Asia, the disease also severely impacted European chestnuts (*Castanea sativa*). After a century of frustrated efforts, the biological tools and knowledge seem to be in place to make restoration of the American chestnut a real possibility. Critical elements consist of high crossability of American chestnut with Asian congeners; simple inheritance of effective partial resistance; a reliable and consistent screening technique; and sexual precocity enabling short intervals (5–6 years) between breeding cycles. It will nevertheless be a long-term endeavor.

No silviculturally useful variation in resistance was found in native chestnut stands, and early breeding efforts sought to exploit the inherent resistance of chestnuts from Asia, where the disease is endemic. The Chinese chestnut (*C. mollissima*), the most wide-ranging and resistant species, was used as the principal source of resistance. Although significant resistance was observed in F_1 hybrids in early trials, it was not adequate for deployment. Further backcrossing to the Chinese parent could increase resistance, but only exacerbate the problem of undesirable growth and form inherited from the Chinese parent.

However, early analysis of certain F_1 and backcross progenies suggested the possibility of relatively simply inherited resistance. Subsequent quantitative trail loci (QTL) mapping with molecular markers of progeny of an F_2 cross confirmed this, and indicated the presence of two or three partially dominant, independently inherited genes responsible for the phenotypic resistance observed. Resistance was expressed as a marked reduction in canker growth rate following artificial inoculation of stem bark with defined cultures of the pathogen. Now the stage was set for a new breeding approach: backcrossing could be done on the recurrent susceptible American parent, to introgress the few genes for resistance from the *C. mollissima* donor, while continually purging remaining Chinese background genes affecting the desired American phenotype. After three backcross (BC) generations from the F_1, only one-sixteenth of the genome, on average, remains Chinese. Each BC generation is screened in field inoculations, using uniform blight cultures placed in drill holes in the bark. Partial resistance is measured by the area of canker expansion after 9–11 months. After the most resistant offspring are selected in each round, a final cross between two BC3 trees ('BC3 F_2s') with

partial resistance should theoretically result in one of 16 trees having two copies of both resistance genes, making them as resistant as the Chinese parents in their pedigree.

Diversity for local adaptation will be provided by including at least 20 different American parents in the BC3 breeding population for any given area. This model, currently being tested and implemented in Virginia, could eventually be applied to a large portion of American chestnut's range. It would involve pollinating American chestnut flowers on sprouting stumps in the wild to produce F_1 seed before they become lethally infected.

Dutch Elm Disease

The impact of Dutch elm disease on American elms has been almost as severe as chestnut blight on American chestnut, and has affected many more species worldwide. It is a more difficult and insidious disease to deal with, because of its complexity. It is a systemic vascular wilt, caused by a species complex of at least two fungal pathogens (*Ophiostoma ulmi*, *O. novo-ulmi*) from uncertain origin in Asia, each with different races spanning a wide range of virulence; it has a saprophytic stage, extending survival of the fungus beyond the disease cycle; it is vectored by several different species of elm bark beetles (Scolytidae), with all the problems of host preference, resistance, and environmental interactions that they entail; and resistance mechanisms and inheritance are difficult to assess and interpret. It is a breeder's nightmare, and were it not for the extremely high aesthetic and amenity value of some elm species for urban and landscape forestry, the nearly century-long effort to develop resistance might never have been attempted. Success has been elusive in both North America and Europe, yet some significant progress has been made.

The American elm (*Ulmus americana*) is the most susceptible of over 40 species in the genus, and over 40 million trees are estimated to have been killed by the disease, including 70% of landscape elms. Sources of resistance are abundant in Asia, especially in populations of *U. pumilla*, *U. parviflora*, and *U. japonica*. However, the relative ease with which resistance genes can be introgressed from Asian chestnuts into American chestnut does not apply to the corresponding elms, because *U. americana* is tetraploid, resulting in severe barriers to breeding with potential Asian donors. Additionally, Asian species tend to be highly susceptible to other diseases and insects, can be climatically maladapted, and generally lack the irreplaceable vaselike architecture that defines American elm. Nevertheless, a few promising *U. parviflora* × *U. americana* hybrids have been made in the University of Wisconsin program that seem to combine resistance traits of the Asian parent with morphological features of the American. Many more such hybrids from diverse sources will be needed for a successful breeding program, but these results are encouraging.

Like the experience with chestnut blight, early selection and testing of American elm were disappointing. Of many thousands of candidate phenotypes tested, only a handful survived for further breeding. These are usually referred to as having tolerance, rather than resistance, and perhaps only 1 in 100 000 American elms have this in useful amounts. To efficiently screen selections, the University of Wisconsin program uses artificially inoculated seedlings or rooted cuttings and measures the volume of discoloration in stems as an index of relative tolerance. Seedlings with less than 50% discoloration, with or without foliar symptoms, usually correlate well with field performance. The criterion for acceptable tolerance in the field was $<20\%$ crown damage. A few long-term survivors, intercrossed with each other and susceptible controls, have produced up to 50% of offspring with less than 20% crown damage after 1 year in the field. Recent results of tests conducted by the National Arboretum showed the ability of a few earlier as well as new selections to respond and then recover from heavy artificial inoculation in the field, equaling performance of some non-American tolerant clones. This suggests that extended evaluation of candidates in the field may be more rewarding in the long term than relying completely on artificial inoculation.

White Pine Blister Rust

The nine white pine species native to North America are highly susceptible to blister rust, and most have been severely damaged in parts of their native range since the disease was introduced over a century ago. All species have important ecological functions, but breeding has been focused on the tall timber species, western white pine (*Pinus monticola*) and sugar pine (*P. lambertiana*).

Unlike most other exotic diseases, a surprising number of resistance mechanisms have been found in white pines to blister rust, even though hosts and pathogen have not coevolved. Three general types of resistance are recognized:

1. MGR, causing classic hypersensitive necrosis in needles on challenge by the pathogen. R genes have been found in several white pines, and exist in a gene-for-gene relationship with cognate genes

for avirulence in the rust. Thus, virulence gene *vcr1* in *Cronartium ribicola* neutralizes resistance gene *Cr1* in sugar pine but not *Cr2* in western white pine, and vice versa for *vcr2*. Single recessive genes are also thought to contribute high levels of resistance in western white pine.

2. PR has two main components in white pines: reduced infection frequency, and different kinds of bark reactions that abort infections after they establish in stem tissues. These mechanisms are more complexly inherited, but can still give effective, if incomplete, resistance. Most important, they are not specifically vulnerable to major virulence genes in the rust.
3. OGR is recognized in adult trees that are free of rust when surrounded by heavily infected cohorts, either in natural stands or seed orchards, but which produce highly susceptible offspring. It is genotype specific, and appears to be very strong and stable. OGR would be useful in stabilizing a crop in later parts of a rotation, but is the least understood and probably most difficult of all the mechanisms to develop.

The problem for breeding is how to recognize, concentrate, and deploy these different mechanisms and the genes that control them into synthetic populations to effect durable resistance. Dominant R genes are easy to breed with and confer immunity to all rust biotypes that lack specific virulence to them. But they place severe selection pressure on the rust for these virulent mutants, which are usually rare in natural populations. When this happens, the selected virulence alleles increase exponentially in frequency in the ambient inoculum, nullifying the protection conferred by populations deployed with only R gene resistance. A strategy devised for sugar pine is to prevent or dampen sudden increases in virulence gene frequency by buffering MGR with different PR mechanisms. This is accomplished in a two-stage process. Seedlings are screened first for MGR by artificial inoculation. Those selected are then outplanted in a field location where the frequency of virulence has been maintained at high levels by continuous natural selection from planted MGR. Since MGR is neutralized in this environment, seedlings surviving have both PR and MGR, and by forward selection will constitute the new parental generation when they mature. Concentration of both MGR and PR genes are effected and increased in subsequent generations (**Figures 1–3**).

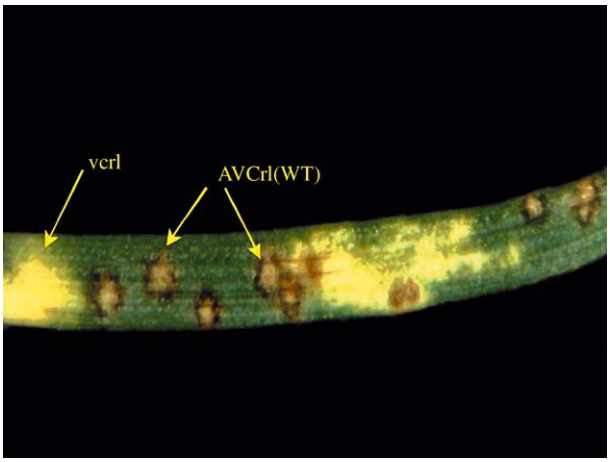

Figure 1 Interaction phenotypes in the white pine–blister rust pathosystem: needle symptoms on sugar pine with the Cr1 gene express as hypersensitive necrotic spots to inoculum carrying avirulence allele AVCr1 (wild type), but as normal yellow spots to inoculum with virulence allele vcr1.

Figure 2 Sugar pine families susceptible and resistant to white pine blister rust in a field trial. The resistant family (far right) has the major gene Cr1 in homozygous condition.

Phytophthora Root Rot of Port-Orford Cedar

Phytophthora lateralis was introduced into western North America from an unknown source over half a century ago, and has since caused widespread mortality in Port-Orford cedar (*Chamaecyparis lawsonia*), a narrow but valuable endemic of northwest California and southwest Oregon. Spread by motile zoospores is rapid and unstoppable – by water coursing through infested stands, or carried by vehicles, people, or animals passing through. Roots are the main infection courts, but foliage can also serve. Environmental variance is high, with low areas being most vulnerable, but root grafts extend infection foci uphill more gradually. The resulting pattern is a mosaic, making assessment of phenotypic resistance ambiguous and difficult.

Figure 3 Partial resistance in sugar pine to white pine blister rust. (a–c) Bark reactions of varying size; (d) low infection frequency. Both trees have Cr1, but have been exposed to natural inoculum with a high frequency of vcr1. b–d reproduced with permission from Kinloch BB Jr. and Davis D (1996) Mechanisms and inheritance of blister rust resistance in sugar pine. In: Kinloch BB Jr., Marosy M, and Huddleston M (eds) *Sugar Pine: Status, Values, and Role in Ecosystems*, pp. 125–132. Davis, CA: University of California, Division of Agriculture and Natural Resources.

Yet, this seemingly intractable problem has recently been overcome by exploiting Port-Orford cedar's unusual reproductive characteristics: precocious flowering (2–4 years) enables rapid turnover of generations compared with other conifers; breeding can be done in containerized orchards in greenhouses; seed is produced abundantly in the same season as pollination; and ease of vegetative propagation by rooted cuttings, even of older trees, permits establishment of ramets of selected candidates, or rapid multiplication of selected progeny genotypes.

Efficient screening is accomplished in two stages. Candidate trees are prescreened by dipping detached branches into zoospore suspensions, then measuring the length of the lesion formed under the bark after a few weeks. The most promising candidates are then rooted, dip inoculated in the same way, planted in nursery beds, and monitored for survival. Most mortality occurs within a year.

The high throughput enabled by these techniques has uncovered rare but highly effective resistance. Although the exact mechanism is not known, progeny of controlled crosses among a few highly selected parents have shown Mendelian segregation of healthy/living : dead offspring, implicating a single dominant gene for resistance. This will greatly facilitate deploying resistant genotypes for restoration of this species. Whether or not the pathogen harbors virulence capable of overcoming this resistance is unknown, but its overall diversity, based on molecular markers, is very low. Partial resistance or tolerance may also be found in the host that could be combined with MGR to mitigate the effect of wider virulence arising. The distribution of MGR is not known, and its rarity could be an impediment to restoring locally adapted, resistant Port-Orford cedar throughout its range. However, the ability to rapidly turnover breeding generations in Port-Orford cedar renders this problem soluble by introgressing nonlocal sources of resistance into local populations through backcrossing.

See also: **Ecology**: Plant-Animal Interactions in Forest Ecosystems. **Entomology**: Bark Beetles; Defoliators; Foliage Feeders in Temperate and Boreal Forests; Population Dynamics of Forest Insects; Sapsuckers. **Pathology**: *Phytophthora* Root Rot of Forest Trees; Diseases of Forest Trees; Insect Associated Tree Diseases; Leaf and Needle Diseases; Pine Wilt and the Pine Wood Nematode; Rust Diseases; Stem Canker Diseases; Vascular Wilt Diseases.

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